
Intra-endodermal interactions are required for pancreatic beta cell induction.

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Public Summary:

Scientific Abstract:

The cellular origin of signals that regulate pancreatic beta cell induction is not clearly defined. Here, we investigate the seeming paradox that Hedgehog/Smoothed signaling functions during gastrulation to promote pancreatic beta cell development in zebrafish, yet has an inhibitory role during later stages of pancreas development in amniotes. Our cell transplantation experiments reveal that in zebrafish, Smoothed function is not required in beta cell precursors. At early somitogenesis stages, when the zebrafish endoderm first forms a sheet, pancreatic beta cell precursors lie closest to the midline; however, the requirement for Smoothed lies in their lateral neighbors, which ultimately give rise to the exocrine pancreas and intestine. Thus, pancreatic beta cell induction requires Smoothed function cell-nonautonomously during gastrulation, to allow subsequent intra-endodermal interactions. These results clarify the function of Hedgehog signaling in pancreas development, identify an unexpected cellular source of factors that regulate beta cell specification, and uncover complex patterning and signaling interactions within the endoderm.

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